Alerts, Notices, and Case Reports

Lung Abscess Causing Horner's Syndrome

DAVID C. DUGDALE, MD
Steilacoom, Washington
KENNETH J. RITTER, MD
DAVID E. WILHYDE, MD
Tacoma, Washington

WHEN FEVER, foul-smelling sputum, and a lung mass with an air-fluid level occur, the diagnosis of a lung abscess is straightforward. In the absence of typical clinical features, the diagnosis may be more difficult. The coexistence of a solid lung mass with Horner's syndrome is characteristic of a superior sulcus (Pancoast's) tumor; the presence of hoarseness strongly suggests recurrent laryngeal nerve compression due to primary or metastatic tumor. We report the case of a patient with a right lung mass, Horner's syndrome, and vocal cord paralysis due to a large lung abscess.

Report of a Case

The patient, a 67-year-old man, noted that for four weeks he had had right shoulder and neck pain, cough, and a 4.5-kg weight loss. Drooping of the right upper eyelid and hoarseness were present for three days. Hemoptysis and night sweats had occurred once but no fever. In the past the patient had had exertional angina and alcohol abuse, and he had a 60-pack-year history of cigarette smoking but no diabetes mellitus.

On physical examination he was afebrile and in no acute distress; he had right ptosis and miosis. A firm, tender mass was present in the right supraclavicular fossa and lower neck. The leukocyte count was 10.6×10^9 per liter without a leftward shift. Chest radiography (Figure 1) showed a 6-cm solid right apical-superior mediastinal mass and leftward tracheal deviation. The initial diagnostic impression was bronchogenic carcinoma with nodal metastases.

At excisional biopsy, the supraclavicular mass was necrotic and contained bloody, purulent fluid. On histologic examination there was an inflammatory response but no malignant cells. Routine culture grew coagulase-negative *Staphylococcus* organisms, but cultures for anaerobes, fungi, and mycobacteria were negative.

Four days later, hemoptysis and bleeding at the biopsy site developed. The patient was afebrile, and a computed tomographic examination of the chest (Figure 2) revealed an 8-cm superior mediastinal mass, without an air-fluid level, that extended into the apex of the right hemithorax. Paratracheal and precarinal adenopathy was present. A cytologic examination of the sputum was negative for malignant cells, and cefoxitin was administered intravenously.

On the second hospital day, fiber-optic bronchoscopy revealed paralysis of the right vocal cord. No endobronchial lesion was seen, but blood was draining from the apical and posterior segments of the right upper lobe. A transbronchial biopsy of the mass yielded tissue with many leukocytes and rare Gram-positive cocci but without evidence of malignancy. Routine cultures grew normal oral flora but were negative for mycobacteria.

The hemoptysis continued three to four times a day. The patient's temperature was 37.8°C, and on the sixth hospital day a percutaneous needle aspiration and biopsy of the mass were done. The aspirate contained many leukocytes and a few Gram-positive cocci. Routine culture grew coagulase-negative staphylococci, but cultures for mycobacteria were negative. The tissue contained an acute inflammatory response without malignant cells. On the ninth hospital day, a cardiac arrest occurred from which the patient could not be resuscitated.

At autopsy, a 15-cm abscess cavity was present in the right upper lobe that communicated with the segmental bronchus and was contiguous with the neck mass. On histologic examination there was inflammatory tissue with numerous Gram-positive cocci but no malignant cells. Mediastinal adenopathy was present and contiguous with, but not part of, the lung mass. The enlarged lymph nodes had follicular and lymphoid hyperplasia without tumor cells or bacteria.

Discussion

This patient's presentation was strongly suggestive of apical lung carcinoma with nodal metastases. Although several diagnostic studies found evidence only of infection, the many clinicians involved with this patient thought that he had lung cancer. The absence of a malignant lesion despite a thorough and careful postmortem examination of the chest was unexpected.

Lung abscesses may result from bacterial, mycobacterial, fungal, and amebic infections. Although bacterial lung abscesses may complicate the course of bacterial pneumonia or septic emboli, they may occur primarily, and a subacute course with fever, cough, hemoptysis, and weight loss is common. The aspiration of oral flora is the hypothesized mechanism of infection, and 60% of bacterial abscesses are purely anaerobic (often with more than one species) whereas 40% are mixed aerobic and anaerobic infections.[1] Although the only bacteria identified in the patient's abscess was a coagulase-negative *Staphylococcus* species, it is likely that this was merely part of a mixed bacterial infection. The hallmark of a lung abscess, a mass with an air-fluid level, is present when communication with the airway occurs but may also be seen with other conditions, including a cavitating tumor. Although the effects of compression, such as tracheal deviation and nerve palsies, have been noted in mycobacterial disease of the lung and mediastinum,[2-4] they are not associated with benign bacterial lung abscesses.

Horner's syndrome, when completely expressed, includes ptosis, miosis, and ipsilateral facial anhidrosis.[2] In the three-neuron sympathetic pathway, the first neurons originate in the hypothalamus, traverse the brain stem, and synapse with the second neurons in the lower cervical and upper thoracic spinal cord. The fibers of the second neurons leave the spinal cord through the first and second thoracic roots (T-1 and T-2), enter the sympathetic trunk, and synapse with the third neurons in the superior cervical ganglion.[5(p342)] The postganglionic fibers follow the branches of the carotid and ophthalmic arteries. Miosis is caused by decreased activity of the pupillary dilators, and ptosis

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From the McNeil Island Corrections Center, Steilacoom (Dr Dugdale), and the Departments of Surgery (Dr Ritter) and Pathology (Dr Wilhyde), St. Joseph's Hospital, Tacoma.

Reprint requests to David C. Dugdale, MD, PO Box 900, Steilacoom, WA 98388.

results from relaxation of the tarsal muscles. In a large series of patients with Horner's syndrome,[2] 88% of those whose cause was determined had lesions of the sympathetic trunk or the T-1 and T-2 nerve roots. Tumors caused 36% of cases, trauma caused 29%, and, in 24%, no cause was ascertained. Of the cases caused by tumors, 75% were due to malignant tumors, half of which were bronchogenic carcinomas. Compression of the T-1 and T-2 nerve roots in the brachial plexus may be associated with radicular pain, weakness, and sensory loss.[6] Of these symptoms, our patient had only shoulder pain, but the location of the mass would permit T-1 and T-2 root or sympathetic trunk compression.

This patient had hoarseness due to right vocal cord paralysis caused by compression of the right recurrent laryngeal nerve. The right recurrent laryngeal nerve turns superiorly around the right subclavian artery, but the left recurrent laryngeal nerve travels more inferiorly and turns around the aorta at the ligamentum arteriosum. Cervical, thoracic, and mediastinal lesions may compress a recurrent laryngeal nerve and cause hoarseness. Malignant neoplasms, trauma, and surgical procedures of the neck account for the majority of cases of vocal cord paralysis.[3-5] As many as 39% of cases are idiopathic, most of which resolve in six months.[7]

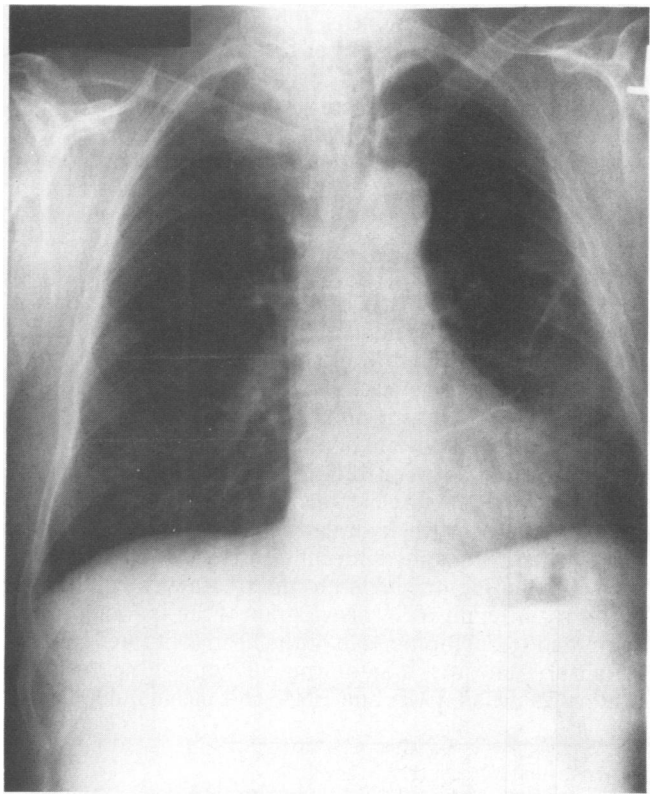

Figure 1.—A posteroanterior radiograph of the chest shows a right apical-superior mediastinal mass and tracheal deviation to the left.

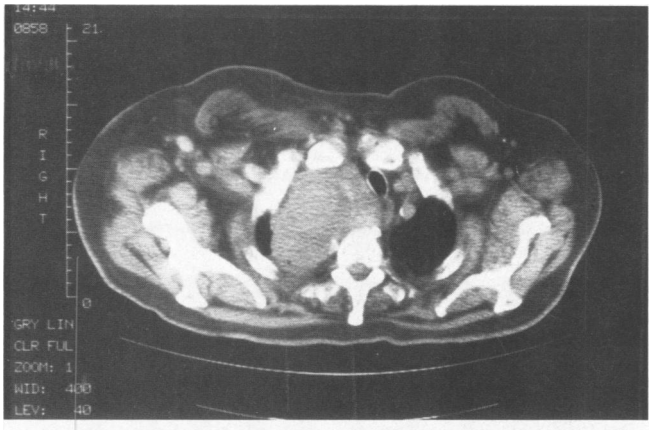

Figure 2.—A computed tomographic scan of the upper chest shows that the mass is inhomogeneous, but no air-fluid level is present.

This patient presented with clinical and radiographic abnormalities usually associated with a Pancoast's tumor. A workup did not suggest a primary lung infection. Diagnostic tests yielded either inflammatory or necrotic tissue, which was assumed to be due to a malignant tumor. The presentation of a lung abscess in this manner has not been reported, even in older series,[8,9] but must be considered during patient evaluation and treatment.

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Transient Infantile Hypertension

NATHAN KUPPERMANN, MD
SUDHIR ANAND, MB, BS
Torrance, California

THE PREVALENCE OF SYSTEMIC HYPERTENSION in childhood is estimated to range between 1% and 5% depending on the criteria used to define the disease.[1-3] Although hypertension in infants and young children is generally considered secondary, a few authors have described hypertension in infants in whom no cause could be determined (essential hypertension).[3,4] This report describes the cases of two infants in whom significant hypertension was noted incidentally before 6 months of age, and no cause was discovered. Both required antihypertensive medication for several months until their hypertension resolved.

Report of Cases

Case 1

The patient, a 35-month-old male infant, was first noted to be hypertensive at the age of 3 months. He was born prematurely, with a birth weight of 1,760 grams. A mild respiratory distress syndrome developed, but he did not require intubation or umbilical artery catheterization. Sev-

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From the Department of Pediatrics, Harbor-UCLA Medical Center, Torrance, California (Drs Kuppermann and Anand), and the Department of Pediatrics, Charles R. Drew-Martin Luther King Jr Medical Center, Los Angeles (Dr Anand).

Reprint requests to Nathan Kuppermann, MD, Department of Pediatrics, Harbor-UCLA Medical Center, 1000 W Carson St, Torrance, CA 90509.